JOINT MEETING

OF THE

NEW YORK NEUROLOGICAL SOCIETY

AND THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

Held in Philadelphia, Nov. 24, 1906.

The President of the Philadelphia Neurological Society, DR. D. J. McCARTHY, in the Chair.

A CASE OF AMNESIA.

By C. W. Burr, M.D.

A man, 55 years old, was found by the police, confused, in the street, in November, 1900. He was taken to the Philadelphia General Hospital, where he had an attack of excitement and violence lasting a couple of days. Since then he has had no recollection of his past life for some years previous, and cannot remember anything that happens now even for a few minutes. The events of his childhood, youth and early manhood and the things that he learned at school he remembers fairly well. He knows that he has been married, but does not know whether his wife is living or dead, and says he has forgotten entirely what she looks like. When he is spoken to about his wife he becomes emotional, but in a few minutes forgets all about it. He knows who he is and has never had any alteration in personality. He is neat and clean in his habits, quiet in manner and shows no moral defect. He knows that he has lost his memory and knows that it is on account of disease. His intellectual judgment is much better than is usually found in senile or presenile dementia. His loss of memory is so great that if he reads (and he reads well) a paragraph in a newspaper he forgets the first sentence before he has read the last. Though he has been told many times that he is in the Philadelphia General Hospital he can never remember it. When in the ward he can reason out that he is in a hospital on account of the number of beds and the presence of people manifestly sick. When he is in the hospital office, however, he reasons out that he must be in some business house on account of the number of desks and clerks. He cannot tell five minutes after a meal whether he has had a meal or not, but reasons that since he is not hungry he must have been fed. He does not know the year, the month or the season of the year. He remembers the year of his birth, but does not know how old he is because, as he says, "I do not know what year this is." In the last few months a few things have made some impression on him, for he now vaguely realizes all the time that he is being treated in a hospital for his sickness.

Dr. B. Sachs said the condition was an extremely interesting one, and the only cases that he has seen that have been equally puzzling have been cases entirely different from the one presented. Two years ago he saw a man who developed an amnesia which was as complete as that of the patient presented, and even more complete. He lost memory of his own personality. He developed this amnesia as an early symptom of general paresis. A few weeks later he developed general paresis with very characteristic symptoms and passed more than a year as an inmate of an asylum. Dr. Burr's case has lasted so long that there is no question of this sort. The difficulty in the class of cases Dr. Sachs spoke of and in the one he cited as an example was to decide whether the man was simulating. In his case there was a law suit pending and he thought the patient had good reason for forgetting his personality and for simulating, but the later developments proved that he was not shamming at all. He is now very near the final stage.

Dr. Morton Prince called attention to the fact that there were two types of continuous amnesia, one the functional form and the other the organic. Charcot was the first to call attention to the functional variety in the famous case of Madame D., who as a result of an emotional shock was found to continuously lose all memory of every experience as fast as it occurred. It was found, however, that the memories were not absolutely lost, but only dissociated, for the memories of her life were rehearsed in her dreams and were completely recovered in hypnosis.

In the organic type, that is in continuous amnesia depending on organic disease, the memories seem to be completely lost, or at least it has not been shown that they were simply dissociated and could be recovered. This form of amnesia has been frequently observed in Korsakoff's psychosis and in alcoholic neuritis. The speaker had observed one very extreme case following what was believed to be from the history of the latter disease, and also in a case that clinically appeared to be one of multiple sclerosis. As to the pathology of this organic form of continuous amnesia we know practically nothing. It is curious to note how a person with a marked continuous amnesia can get along without being markedly incommoded and without allowing it to be suspected. The case of multiple sclerosis, for example, had attended the hospital clinic for three months without the amnesia being suspected.

A CASE OF COMBINED POSTERIOR AND LATERAL SCLEROSIS, WITH INVOLVEMENT OF THE CELLS OF THE ANTERIOR HORN.

By J. W. McConnell, M. D.

Dr. Joseph Collins said he did not know whether it was the intention of Dr. McConnell to present this case as a new type of disease, but, if so, he was loth to accept it as such. Sufficient had been said about its occurrence and about the character of the disease to make it clear that it was a manifestation of diffuse syphilitic disease of the cord analogous to that Nonne had described in his last contribution. He could not say at the moment whether he had seen more than two or three cases similar to this or not, but he was quite sure he had seen that number. He understood that there were some sensory disturbances in Dr. McConnell's case. There must have been widespread, more or less disseminated, lesions throughout the spinal cord which entitled it to be classed as a disseminated syphilitic lesion.

Dr. C. E. Atwood saw one case of subacute combined sclerosis when an assistant of Gowers, and it corresponded very closely to this, but it was not so far advanced. It had similar paresthesia in various parts of the body, especially about the anus and genitalia and in the extremities. The age of the patient was about the same and there was, as in this case, a good deal of anemia. He believed that in these cases one of the earliest symptoms is a very pronounced anemia. There was in his case a condition of spastic paraplegia with anesthesia, girdle sensation, increased knee jerks, Babinski, and inability to walk, as the disease advanced, from weakness and marked spasticity. The disease runs a rapid course and on autopsy marked wasting of the white matter in the cord is found. The disease may be due to some chemical toxic substance in the blood.

Dr. McConnell, closing, said he had not stated that these patients had cranial symptoms. One of the patients showed Argyll-Robertson pupil. He had not intended to describe any new clinical type, but it had occurred to him often that the appellation cerebro-spinal syphilis is such an indefinite one that we should have a better term. Other scleroses were spoken of as posterior sclerosis, combined sclerosis, lateral sclerosis. Why not give a clinical pathological diagnosis to our cerebrospinal syphilitics?

A CASE OF TABES IN A NEGRESS.

By John K. Mitchell, M.D.

Dr. Mitchell said he was sorry to say that he had just learned that the patient whom he had hoped to show had at the last moment refused to come to the College of Physicians, and he would therefore say only a very few words about her.

The only unusual features of the case and the reason why he wanted to exhibit her lay in the rarity of the occurrence of locomotor ataxia in a black of pure blood. In spite of the frequency of occurrence of syphilis among the blacks, ataxia is so rare in his experience as to be almost unknown, except in those who obviously have a greater or less share of white blood. It is probable, of course, that there are not a great many negroes in the country who have not some percentage of white blood, but so far as one can judge by appearance this ought to be one of the certainties, and the woman herself is entirely ignorant of any trace of Caucasian in her pedigree.

She has a Charcot joint in both knees and in one ankle. She has been under Dr. Mitchell's observation with the disease for three or four years. She is pretty nearly at a standstill and presents no symptoms of any peculiar interest.

Dr. S. Weir Mitchell regretted very much that his son could not present the case. There were three disorders of the nervous system which the negro escapes. This was certainly one. He thought chorea, again, was another. Chorea among pure black children was practically unknown in this country and Cuba, and he has had occasion recently to find from inquiries sent out that this was probably the case as concerns exophthalmic goiter. He found to his surprise that in Cuba it is exceptional and rare among the negroes. Physicians wrote him from Cuba that it is scarcely heard of among the pure-blooded blacks. These were things which the negro escapes.

Dr. J. A. Booth had seen six cases of tabes in the colored race, and they were all patients in the Colored Home in New York City. Among the six there was no question of four of them being of the pure colored race. He had also seen one case of chorea in the Colored Home.

Dr. L. Pierce Clark said he had reported in his second thesis on myoclonus-epilepsy a full-blooded negro patient who died of myoclonus-epilepsy. The case had been first diagnosticated as choreic epilepsy before admission to the Craig Colony for Epileptics, but the movements were typical of myoclonus as seen in the association disease. In an analysis of two thousand cases of idiopathic epilepsy in his service at the colony there were at least six full-blooded negro patients, about the normal ratio existing between the races in the general population of the State.

Dr. H. M. Thomas said that in Baltimore were seen a considerable number of cases of tabes in black people, but he found it very hard to prove that they were pure-blooded blacks. He was sorry his statistics were not perfectly accurate on that account. Any person who had any negro blood was classed as black, and they came in among these cases. Many of them did not know their parentage and he had to judge by looks.

Dr. J. K. Mitchell said that it was difficult to be sure in any given case of negro that he had no white blood. He supposed there were few negroes in this country without any *taint* of white blood. This woman looked as if she had none, and when asked she said: "Oh, Gawd, I don't think so, both my grandmothers was mos' 'spectable women." Considering the frequency of syphilis among the blacks, the rarity of tabes must be very great. There are 70,000 negroes of varying degrees of darkness in Philadelphia alone, it was remarkable that no more cases should turn up at the nervous clinics, the case under discussion being absolutely the unique instance known to Dr. Mitchell of ataxia in a negro of presumably undiluted African blood.

A CASE OF PROBABLE APRAXIA.

By John H. W. Rhein, M.D.

Dr. Rhein stated that the man was fifty-five years of age and had been admitted to the Philadelphia Home for Incurables on July, 1906, complaining of loss of vision. The family history, as well as the previous history, was practically negative. Three years ago he began to lose his eyesight, and this loss progressed gradually until at the end of two years he was unable to read. At present he is unable to see anything but a bright light, and is not always sure of this. He has entirely lost his ability to recognize the position of his body and limbs. He cannot tell whether he is standing or sitting, or what position his arms or legs are in. He has lost entirely his sense of localization. He recognizes a light touch in certain places, but cannot locate it. When asked to place his right hand upon his nose or ear he places his hand upon his knees or his chest, stating at the same time that he is touching his ear or nose. When he is asked to squeeze the hand of the examiner with his left hand, he grasps his right leg with his right hand and squeezes, believing that he is squeezing the examiner's hand with his left hand. At the same time he does not move the fingers of his left hand whatever. When a watch is placed in his right hand, and he is told to place it to his ear, he does it frequently correctly, but some times puts it to his mouth. He does not recognize the ticking of a watch, but when asked if he knows what the object is he replies: "Yes, it is a watch." He states that he knows this because his reason tells him that the watch would be the object which the examiner would place to his ear to test his hearing. The tactile sense in his left hand appears to be lost; in the right hand it is present. Tests of the pain sense were rather unsatisfactory because they irritated the patient very much, and obscured the accuracy of the observations. It is probable that this sense is retained in both hands. He is unable to distinguish between light and deep pressure. He recognizes the difference between heat and cold in the right hand, but does not always recognize the difference in the left hand.

Dr. Neinan, the resident, found the thermal sense markedly impaired all over the body. The stereognostic conception is impaired; he does not recognize the nature of objects placed in his hands. His gait is "pattering," he takes small steps, first with the right foot, and usually toward the right.

In a brightly lighted room, if a hand is placed before his eyes, while he is walking, he turns away from the hand without recognizing that an object is placed before his eyes. He does not recognize objects in a brightly lighted room, and will frequently bump into a table or the wall in walking alone. Both arms and legs are rigid, especially on the left side, although not truly spastic. The knee jerks are increased; there is no clonus or Babinski. The arm jerk is present on both sides. The station with the feet together is good. The grasp is good and equal on both sides. The tongue is protruded straight in the median line, and is tremulous and tooth-indented. The taste sense is markedly altered, while the olfactory sense seems practically unimpaired. He cannot write his name, and in making this effort makes a scrawl, writing from the right to the left.

Examination of the eyes by Dr. W. C. Posey revealed the early changes of simple atrophy of the optic nerves. There was no Wernicke's pupillary inaction and no palsy of the extraocular muscles. The pupils reacted to light. Dr. W. G. B. Harland reported that there was nothing in the examination of the ears that would not be found in an old man with mild middle-ear disease.

The patient is intelligent, answers questions promptly, and his memory appears to be good. He is well nourished, his appetite is good, his digestion unimpaired, and his bowels regular. He complains of nothing excepting his blindness. He has a quick temper, and becomes frantic when the examination is pushed beyond a certain point.

The case bears some resemblance to those cases of apraxia which have been reported. However, a further study of the case must be made before a diagnosis can be ventured upon. At present this much can be said, that bilateral lesions involving the occipital and parietal lobes would explain most of the symptoms which this patient presents.

Dr. A. Fraenkel, of Haiden, Switzerland, said that he imagined the case might have been one of functional disease, but as he saw the patient coming in, he was sure it was not functional, but that it was a sort of bulbar disease, and that it interested us to know whether he had any ophthalmological history. Dr. Rhein stated that he had very early stages of optic atrophy. This would not explain his blindness. There might be a double lesion in the occiput with symptoms of apraxia.

Dr. Joseph Collins said that those who attended the American Neurological Association would recall the type of disease which he had attempted to portray as a clinical entity, a discussion of which had appeared in THE JOURNAL OF NERVOUS AND MENTAL DISEASE. They would recall that the most striking characteristic of the disease was the alteration of the patient's appearance. The individual became transformed from a person expressing grace in movement, and relaxation in repose into an immobile, inanimate replica of the normal person. The second was that the cases first impress one as being paralysis agitans, and that when you came to dissect and analyze the one symptom above all was the immobility. There was no motorial function of the body which might display the disorder. This immobolization gives a characteristic attitude and gait, and to a lesser degree a characteristic physiognomy. The gait is the most remarkable feature of the patient. The stride is short, oftentimes only a few inches, the feet slightly lifted, the rhythm generally slow, occasionally rapid. One case he had complained of impaired vision. The disease was clean-cut in its delineation, quite as much so as Parkinson's disease. He has had several autopsies, and has had six or seven brains which show the distinctive lesions. What the lesion is it was imposible to describe in three or four minutes. Some changes were confined to the motor areas and others were distributed throughout the entire brain. The disease was that which Charcot probably had in mind when he described abasia trepidant; afterward an attempt was made by a Polish clinician Bieganski to describe it. In hospitals where many patients were undiagnosticated it had been forced upon him that there was a type of disease of which this man is the best exemplification he had ever seen, that has a definite clinical type and which must at the present time be called a definite form of general cerebral arteriosclerosis with special symptoms. The patient now exhibited seemed to him to be a fairly typical example of the disease.

Dr. Joseph Fraenkel said that the cases he had seen did not fully coincide with the description given by Dr. Collins. Those described originally by Charcot under the name of "gait stuttering and stammering" which manifested a condition like the patient presented, he had never seen except once. He thought the same patient was presented in New York. They did not make a diagnosis of his condition then.

Dr. Rhein said that he had not yet had an opportunity of looking into the literature exhaustively, but that there were three cases recorded which were in many respects similar to the one exhibited. One was a case reported by Dr. S. Weir Mitchell, a woman giving a history of gradually increasing blindness, together with loss of the stereognostic sense. She died under Dr. Pershing's care in Chicago, and an autopsy revealed the presence of cysts situated in both occipital regions, and general sclerosis of the cortex. Another case was reported by Dr. Charles W. Burr, without autopsy, in which the patient had progressive blindness and tactile amnesia.

The third case was described by Liepmann under the title of "Unilateral Amnesia." In his case there was degeneration of the occipital and parietal regions, together with portions of the corpus callosum.

Dr. Rhein thought that, as far as he had gone into the study of the case, the idea suggested by Dr. Fraenkel that the lesion was in the occipital lobes, was in part, at least, the best explanation of the case, although he himself inclines to the view that the parietal, as well as the occipital lobes, are involved.

A CASE OF PROBABLE PARALYSIS AGITANS IN A BOY OF TWELVE.

By T. H. Weisenburg, M.D.

The speaker stated that the family history and the past social history of the patient were uninteresting. The boy had the ordinary diseases of childhood, and up to a year ago was considered normal. At this time, while going to school, his teacher noticed that the boy became somewhat stupid and slow mentally, and that he was not as rapid in learning as he had been. The teacher also noticed that his speech became more or less monotonous and indistinct. About the same time the boy began to have a shuffling gait, and every once in awhile would trip over slight objects. The boy at present has a stiff attitude, he holds his body rigidly, head bent over, arms held to his side. When he walks the body is inclined forward, the steps are short, shuffling, and he has a tendency to slip over the slightest object. His face is mask-like, suggesting strongly the facies of paralysis agitans. His speech is slow and monotonous and hard to understand. He has dribbling of saliva almost constantly. He smiles very rarely and he hardly ever cries. His mental condition is slow; he responds to questions fairly well, but it is evident that as compared to a boy of his years and opportunity for education he is undeveloped mentally. There is no apparent coarse tremor, although he has fine tremor of the upper limbs and sometimes of his head. He holds his hands in flexion and somewhat in the position of a case of paralysis agitans, but there is no pill-rolling movement. Any sudden push forward or backward will cause him to fall. There is no festination, although others have observed a tendency to it. The reflexes are increased generally, and sensation is normal.

The patient, from appearance, attitude, facies, tendency to shuffling gait and falling forwards and backwards suggests paralysis agitans. It is rather difficult to say at this early date and in such a young person whether the case is a true one of this character or whether it is a forerunner of some other disease as multiplesclerosis. There is no denying the fact, however, that it strongly suggests paralysis agitans.

Dr. Charles Dana said that about fifteen years ago a boy came into his hands with symptoms very like this case. The boy was about this age when he first came and was finally transferred to the Montefiore Home. He thought the picture had gradually changed to multiple sclerosis. It seemed to him that this was precisely what this boy's condition would be ten years from now.

Dr. Joseph Fraenkel stated that he and Dr. Sachs had had a case of paralysis agitans in which the diagnosis had been made by the children with whom the child played, long before they had made it. The boy's mother had told Dr. Fraenkel that his schoolmates called him "false face." He presented for a number of years the most characteristic picture of paralysis agitans that he had ever seen. The only feature not characteristic of paralysis agitans was nystagmus. Later the patient began to show evidences of disease of the pyramidal tracts; markedly exaggerated tendon reflexes and ankle clonus. Three years ago Dr. Turner, of London, suggested to the speaker the possibility that cases of this kind were some type of Little's disease—cerebral diplegia.

Dr. Sachs said that the case resembled that which Dr. Fraenkel referred to very strongly, but he felt great hesitation in arriving at a diagnosis in a case he had seen for only a few minutes. He thought he was one of the first to see the case that Dr. Fraenkel referred to, and suggested that it was a peculiar fact that when a condition resembling paralysis agitans occurred in a younger individual it should assume the form of multiple sclerosis. Paralysis agitans was much more common in the later periods of life than in the early. All the symptoms here suggested paralysis agitans. It would be interesting as years go on to follow developments in this case. In the boy's face there was a suggestion of scleroderma, but on examination of the hands he thought there was nothing of the sort. We should be careful about establishing new types. It was interesting to note what an unusual number of atypical cases occur which it is not easy to label one way or another.

A CASE OF HEMIATROPHY OF THE FACE.

By A. Gordon, M. D.

The gradual wasting of the muscles followed an attack of two days' duration of neuralgic pain in the infraorbital region on the right side. Now, eight years after the onset, the atrophy is complete and the reaction of degeneration is distinct. At no time were there vasomotor disturbances on the affected side, and the pupils are equal, a fact which militates against the sympathetic origin of the disease in the present case. Dr. Gordon reviewed other theories, criticizing them, and concluded that facial hemiatrophy may be caused by involvement of the lower sympathetic ganglion, by the fifth nerve, by the seventh, by the Gasserian ganglion, finally by a cerebral lesion.

Dr. M. Allen Starr asked if any one present had ever noticed any facial hemiatrophy following operations on the Gasserian ganglion. He had never seen one. In only one case of hemiatrophy had he seen neuralgia symptoms referable to the fifth. He had considered the advisability of exsecting the ganglion in hemiatrophy, but unless it is proven that the fifth ganglion has a relation to hemiatrophy he thought it would be a rather risky operation.

Dr. Spiller, in replying to a question from Dr. Starr, said he had never seen a case of atrophy resulting from operation on the Gasserian ganglion.

Dr. Dana said that this was a typical case of hemifacial atrophy. The atrophy was very slight. It was progressive; that several of the cases he had seen had been associated with scleroderma and this patient's face suggested it. He had observed a number of cases where the Gasserian ganglion had been removed for a long time without any effect upon the trophic condition of the face.

Dr. Morton Prince said that an alternative hypothesis must be considered; namely, whether it was not rather a case of muscular dystrophy rather than of so-called ganglionic hemiatrophy. In most of the cases of hemiatrophy reported the bones and other tissues were involved. In this case this did not seem to be the fact, but the atrophy was limited entirely to the facial muscles which were paretic. He believed therefore that the theory of muscular dystrophy must be entertained, or possibly the case was one of simple facial paralysis.

Dr. Knapp said that his own experience coincided with that of the other gentlemen who have spoken; that he had not found hemiatrophy following lesions of the cervical sympathetic or operations on the fifth nerve. The patients which he had seen had had no paresthesia. This case seemed to him rather peculiar on account of its general diffuse character. In the early stages of hemifacial atrophy he had noted, as others had, the involvement of bone. In one case in particular the trouble began with a marked wasting of the tissues in the forehead and bone as a depressed band right down the forehead close to the medium line with a diffuse wasting of the rest of the face. A general diffuse wasting like this without bony involvement he had not seen in a case of hemiatrophy.

Dr. Sachs said that he was impressed with the fact that this might belong rather to the class of cases spoken of as pseudo-hemiatrophy. There were marked sensory changes in this case, not of syringomyelic type. This case seemed parallel with two or three cases he had observed closely for years in private practice which began with very marked pain within the trigeminal area; they suggested syringomyelitic disorder. In one the marked trigeminal symptoms had been followed by atrophy just as was seen in this case, an atrophy which was muscular rather than an atrophy that involved the subcutaneous tissues, the bone and so on, as was the case in progressive hemiatrophy. These cases he referred to he had not been able to label properly. He rememberd that Dr. Jacoby some years ago thought that they might be incipient cases of tabes, but up to the present time no symptom of tabes had developed. They were surely cases of trigeminal disease no other symptoms following.

Dr. Adolf Meyer asked if there had been any electric reaction of degeneration in the facial nerve.

Dr. Gordon, in closing, replied that Dr. Prince's remark concerning the possibility of a muscular dystrophy cannot be accepted in this case for the reason that the duration of the disease has been eight years and further, the disease is confined to the face. His arms and the muscles of the neck are absolutely intact. The reaction of degeneration is very pronounced. Therefore the duration of the disease for eight years and the lesion being confined exclusively to the face throw out the diagnosis of muscular dystrophy.

As to another possible origin of the disease, Brissaud suggested the possibility of syringomyelia of the medulla. The patient does not present sensory symptoms corresponding with that. There is no vasomotor disturbance so as to make us think of the sympathetic system. As to the question of operation, Dr. Gordon did not see what operation could accomplish under these circumstances.

(To be continued.)

NEW YORK NEUROLOGICAL SOCIETY.

October 2, 1906.

The President *pro tem*, Dr. Adolf Meyer, in the Chair.

A CASE OF SUBCORTICAL TUMOR.

By Richard Weil, M.D.

The patient was a man, forty-two years old, a Russian, who, three months before he came under observation complained of severe headaches from which he could obtain no relief. About the same time, some impairment of the mental faculties was noticed, and there was impairment of vision which rapidly progressed to total blindness.

During the first two months of his illness he also had two convulsions. According to the history given by his wife, he would wake up in the night, complaining that his left arm was shaking. From the arm, the movements would progress to the corresponding leg, and finally the entire left side would become involved, and unconsciousness would supervene.

When the man entered the hospital, the optic nerves showed a secondary atrophy. The physical examination revealed very slight impairment of the motor functions. On the left side, the reflexes were slightly increased; there was no clonus in the patella nor ankle. Power was unimpaired; no ataxia; no Babinski. He had perfect thermal sensibility, and the pain sense was unimpaired. There was only one pronounced symptom, namely, astereognosis, which persisted up to the time of operation, and he was unable to